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Reflex Aural Symptoms without Aural Disease.

Aural Disease Exciting Reflex Symptoms.

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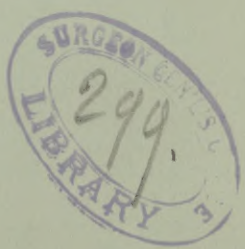

FROM

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES.

OCTOBER, 1886.

Extracted from the
American Journal of the Medical Sciences for October, 1886.

REFLEX AURAL SYMPTOMS WITHOUT AURAL DISEASE. AURAL DISEASE EXCITING REFLEX SYMPTOMS.

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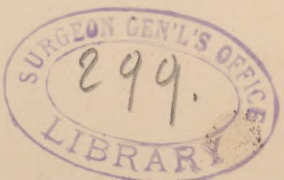
In the *Annales des Maladies de l'Oreille du Larynx et des Organes Connexes*, September, 1884, an article appeared, entitled "Aural Symptoms occurring in Hysteria, and the Hysterical Element in Aural Disease." Two cases were reported: one of aural symptoms simulating inflammation of the mastoid, and caused by hysteria; the other, where slight hysterical symptoms were much increased by inflammation of the middle ear, with serous effusion. The report of these cases, which opens a wide field of research in the domain of hysteria, directs attention to another group of cases, occurring mostly in children, and which apparently is to be classed under the head of aural symptoms arising reflexly from trouble in other parts of the economy, and conversely reflex symptoms of other organs caused by aural disease.

This subject has, as yet, been very imperfectly recorded in the literature of pediatrics, and yet is of considerable importance in the symptomatology, prognosis, and treatment of children's diseases; and in order that our meaning may be thoroughly understood by our readers, we will, before discussing the subject in detail and reporting our cases, present briefly the following examples, illustrating the two branches of the subject:

Case I. *Reflex aural symptoms; no aural disease.*—A. D., aged fourteen months, has eight incisor teeth, and from general appearances is probably in the process of cutting the first four molars; frequently suffers from pain in both ears, partial temporary relief to the pain



being obtained by inflation of the Eustachian tubes; careful examination of the ears reveals nothing abnormal beyond, at times, an evanescent injection of the membrana tympani.

The pain gradually grew less severe, and the attacks less frequent, but the trouble continued until the molars were well up under the gums.

Diagnosis. Irritation of dental nerves in bone sockets; reflex irritation and suspense of vasomotor inhibition through the otic ganglion, causing dilatation of tympanic vessels, pressure, and aural pain.

Case II. *Aural disease; reflex pulmonary symptoms.*—B. C., three and a half years old, has for some months had purulent inflammation of left middle ear; sometimes a discharge being present, and again ceasing for several weeks; but perforation of the membrana tympani always remaining unclosed.

December 24. For some days has been having an offensive purulent discharge from the ear, with a temperature varying from 99° to 101° F.; was out of doors in the morning and exposed to a cold easterly wind; did not seem well in the middle of the day; loss of appetite, chilly sensations and pains in limbs; throat slightly reddened; in the night, symptoms grew worse; the temperature went up to 105°, the pulse to 160, and the respirations to 75; nothing abnormal was found in the lung or larynx, and yet the quickened respirations were of a pulmonary type; examination of the ears showed nothing to account for the severity of the symptoms but the same purulent offensive discharge; temporary relief to the respirations and a reduction of temperature were obtained by inflating the Eustachian tubes and syringing the ear; the symptoms, however, returned on the following day, and when seen by another physician in consultation (respirations 75; temperature 104°; pulse 160) were pronounced to be those of pneumonia, until repeated and careful physical examination proved that the respiratory tract was not involved, and inflation and syringing were followed in a few hours by reduction of the respiration to 25, and the temperature to 98.2°; no further trouble arose in the case.

Diagnosis. Purulent inflammation of middle ear; reflex symptoms simulating pneumonia.

The intimate association of the filaments of the pneumogastric nerve with the ganglia of the sympathetic system, will suffice for the present as an explanation of the marked pulmonary symptoms existing in this case, where the source of the disease was evidently aural; we shall, however, later refer again to these pulmonary symptoms in a case somewhat more complicated than the one just cited; our first example also will be more clearly explained when we come to speak of the connection between the teeth and the ears.

One point especially to be urged in these cases of aural lesion of reflex origin is that the preliminary congestion occurs, in the great majority, in the upper portion of the tympanic cavity, and upper portion of the membrana tympani. The more immediate source of the blood supply of this region is a tympanic branch of the carotid, sometimes two or three branches, passing through openings in that portion of the bony wall of

the carotid canal which forms a part of the anterior inferior wall of the tympanic cavity.

These branches are necessarily short, of considerable size as compared with other sources of blood supply, proceed directly to their point of distribution, and have not, according to Politzer, the usual capillary terminations; these facts, taken in connection with the force of their circulation derived so directly from the carotid, make them especially liable to sudden and extreme dilatation on the suspense of vasomotor inhibition, and that the nervi-vasorum constituting the carotid plexus at this part of its course come largely from the otic ganglion (Woakes), and are hence placed in direct communication with distant parts, is the second of the important factors which explain the sudden and often unexpected implication of the middle ear in the diseases of other organs.

The sympathetic nerves of the lining membrane of the middle ear spring from the sympathetic plexus which accompanies the carotid artery in its canal. By means of orifices in the canal, several small branches of this plexus enter the tympanic cavity as nervi-carotico-tympanales, to form in its anterior portion together with the ramifications of Jacobson's nerve, and the nervus petrosus superficialis minor, the plexus tympanicus. From this proceed the finer nerves for the whole lining membrane of the middle ear.

The important influence exerted by the sympathetic nerves upon the vessels of the tympanic cavity is shown by Prussak's experiments on dogs.[1] The tympanum was opened, and while the portion of the sympathetic in the neck was galvanized, the vessels of the promontory and of the membrana tympani were examined by a magnifying glass. Shortly after the beginning of the irritation, the vessels became smaller, diminishing so as to be almost invisible; when the irritation ceased a considerable expansion of the vessels took place.[2]

We shall confine ourselves, for the sake of brevity, to cases implicating the middle ear, leaving out of consideration that large group of symptoms connected with disease of the internal ear which has already been investigated, and also not attempting to cover the whole ground of reflex middle ear affections, merely citing such illustrative cases as have happened in our practice.

A few words regarding the general mechanism of the phenomena represented in these cases, and so admirably spoken of by Woakes in his article on "Sources of Ear Affections in Infancy and Childhood," may be of aid in fully appreciating the otherwise inextricable complex of symptoms which we are about to describe, and we can then take up the

[1] Prussak. Ueber Anastomosen zwischen den Gefässbezirken des Mittelohrs und des Labyrinths. Archiv f Ohrenheilkunde, xi.

[2] Politzer. Diseases of the Ear. English edition, p. 50.

especial anatomical connections which serve to elucidate the individual case. Woakes says, "The anatomical mechanism, to the consideration of which these remarks lead, will be found in that portion of the nervous system constituted by the ganglia of the sympathetic chain and its afferent and efferent branches."

By far the most important fact in connection with the sympathetic system is that, with one or two exceptions, all sensori-motor nerves include fibres belonging to it. These sympathetic fibrillæ proceed to their companion cerebral or spinal nerve from that ganglion which is nearest to the latter when it issues from the spinal canal, or that join it from ganglia near which it passes in its course.

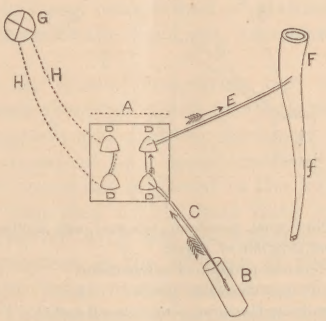
These sympathetic fibres are afferent in their function as regards the ganglion—*i. e.*, they convey impressions from the tissues to which they are distributed to the ganglion, or beyond it, to centres within the spinal cord. They may be looked on as taking this ganglion in their course to the general vasomotor centre, hypothetically seated in the medulla oblongata, but it must not be forgotten that when these fibres thus enter a ganglion, they communicate with its caudate cells. This important fact brings them into communication with another set of nerves coming from very different directions to the same ganglion.

The second set of fibrillæ referred to as entering the ganglion, proceed by a similar course from the general centre, to join a given sympathetic ganglion; they then, similarly to the afferent sympathetic fibrillæ, mingle with the caudate cells of the ganglion, after which they quit it to seek their several destinations on the coats of the arteries. They are efferent or centrifugal in their function, conveying impressions from the general centre or the sub-centre constituted by their ganglion, to the arteries whose calibre it is their function to regulate; hence they are usually called vasomotor nerves. It will probably be found a not incorrect inference to regard these centres, whether primary or secondary, as playing a subservient part to the tissues which animate them. From this point of view, they will be reduced to the level of stations for receiving and transmitting impressions originating in the vital work of the areas with which they communicate, and, therefore, devoid of any power of originating such impressions, though they do undoubtedly modify the impulses passing through them, according as their vigor or vital energy is less or more than normal. We may conclude then that the sympathetic ganglia not only play the part of secondary centres or sub-centres, receiving and transmitting impressions quite independently of the general centre, but that they are also correlating organs, by means of which afferent tissue impressions from one direction are reflexly referred to a totally different tract, where they find expression as modifications of vessel calibre in that tract, the afferent impressions being

manifested through their medium as efferent impulses in the area to which they are thus reflected. In other words, the sympathetic ganglia are to be regarded as so many stations situated on the lines traversed by vasomotor impressions, in which the "points," so to speak, are managed and by means of which impulses are transferred from one line to another.

The following diagram will illustrate, in a general way, the foregoing remarks.

FIG 1.



A, sympathetic ganglion.
B, sensori-motor nerve.
C, afferent sympathetic fibres from sheath of B.
D, caudate cells.
E, efferent sympathetic fibres proceeding to artery F.
F, artery dilated.
f, normal size of artery beyond the sympathetic influence.
G, general vasomotor centre.
HH, the dotted lines indicating the course of the fibres forming the roots of the ganglion in the spinal cord to the general vasomotor centre G.

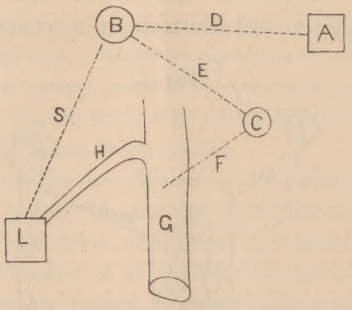
We can now report a class of cases which represent the reflex connection between the ear and the larynx, and we will first indicate simply, by means of Fig. 2, the anatomical relations between the organs.

The irritation of the sensitive fibres of the auriculo-pneumogastricus distributed in the meatus, and to the membrana tympani, is reflected along the motor fibres of the superior laryngeal nerve, exciting in the larynx the act of coughing, by causing contraction of the crico-thyroid muscle. Now, where the original irritant, either in the meatus or in the membrana tympani, by its continued presence involves the vasomotor fibres associated with the auricular nerve, they conduct their impression to the ganglion of the pneumogastric, and thence it is deflected through a sympathetic fasciculus proceeding from it to the first cervical ganglion, this again through the nervi molles carries the impression to the external carotid artery and by its branches to the mucous membrane of the larynx, and as a result of reflected vasodilator impressions we may have congestion of the vessels supplying the mucous membrane of the larynx and perhaps effusion from these vessels.

*

As examples of reflex irritation, having their origin in this way, may be mentioned the hoarseness which sometimes accompanies the impaction of cerumen, and which disappears almost immediately after the removal of the mass, and the case of a young girl who was relieved of a

FIG. 2.



A, auditory canal, membrana tympani, and middle ear.
B, second ganglion of vagus.
C, first cervical ganglion of sympathetic.
D, auriculo-pneumogastric nerve.
E, sympathetic fasciculus connecting B and C.
F, nervi molles vasomotor connection with external carotid.
G, external carotid.
H, laryngeal artery.
S, superior laryngeal nerve.
L, larynx.

persistent laryngeal cough of several months' duration, accompanied by excessive expectoration, producing extreme emaciation, by the removal of a bead from the external auditory canal. The reverse of these laryngeal symptoms reflex from the ear, is represented by that class of cases noticed by Tröltsch and others, in which long-standing laryngeal disease is followed by deafness, and Gerhardt's case of severe pain in the ear, apparently depending on ulcerative destruction of the epiglottis.

The following is an interesting case of middle ear disease causing reflex laryngeal cough.

CASE III.—A child, five years old, who had been under our care for various diseases, and had developed purulent inflammation of the middle ear, was noticed, when under treatment for the otitis, to have a spasmodic, apparently laryngeal, cough, not to be accounted for by any abnormal condition of the air-passages. Whenever the discharge was checked, the cough was persistent, and resisted all the remedies which were employed to control it, but always grew less and frequently disappeared entirely, whenever the ear began to discharge again. This sequence of symptoms happened so frequently that there seemed to be no doubt that there was a connection of dependence between the discharge and the cough, and many an amusing appeal was made by the writers of this paper to each other, to allow the especial part to which they were directing their remedies, to remain quiescent, which only

ended by the baffled physicians sending the despairing parents abroad with the child, to a climate where a more complete cure could take place in the ear.

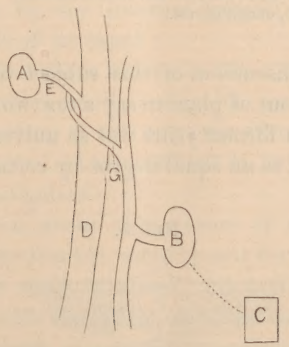
We will next report the reversal of the impressions conveyed by the filaments of the pneumogastric and the allied sympathetic fibrillæ, occurring in the second case described in this paper, in which aural disease was followed by pulmonary symptoms. This case is an illustration of the primary implication of the middle ear in pulmonary tuberculosis.

Case IV.—A young man, twenty-three years of age, with marked tuberculosis of the right lung, complained of tinnitus aurium, and especially of a sense of fulness in the right ear, accompanied by decided impairment of hearing.

Examination of the ear showed congestion and œdema of the upper portion of the membrana tympani which was followed by perforation of the membrane of Shrapnell, and a serous discharge which soon became purulent, and was accompanied by a rapid breaking down of the tissues. A week later the same symptoms occurred in the left ear, with the same appearances, and the transition within four days to a well-established purulent otorrhœa. The left lung was also affected. In this, as in similar cases, a marked symptom is the absence of the severe pain which usually accompanies the inflammatory affections of the middle ear and membrana tympani. The anatomical connection of the parts is obviously through the pneumogastric ganglion B, Fig. 2, by means of the auriculo-pneumogastric nerve.

We can now, guided by the principles which we have above endeavored to elucidate, return to the study of those especial ear cases illustrated

Fig. 3.



A, tympanic cavity.
B, otic ganglion.
C, tooth.
D, internal carotid.
E, tympanal branch.
F, auriculo-temporal nerve.
G, auricular branch of auriculo-temporal nerve.

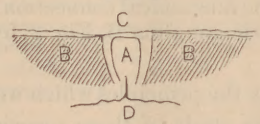
by the first case reported in this paper, in which there is congestion of the auditory structure arising from the irritation of teething, and we have

to ascertain how a congestion of the ear sufficient to give rise to severe pain can be caused in a reflex way by irritation connected with the teeth and gums, for clinically the occurrence of this phenomenon is quite frequently met with in infants during the dental periods. Woakes says:

"A considerable portion of the blood supply of the membrane of the drum is derived from an artery that leaves the internal carotid in the carotid canal and proceeds by a very short course directly to its destination. Being thus closely connected with a large arterial trunk, this small tympanal branch is very favorably situated for a speedy augmentation of its blood supply. Now the nervi-vasorum constituting the carotid plexus at this part of its course come largely from the otic ganglion. On the other hand, the inferior dental nerve supplying gums and teeth also communicates with this ganglion."

We thus arrive at a direct channel of nerve communication between the source of irritation, the tooth, and the vascular supply of the drum-head. The vessels of the membrana tympani become largely distended, and the attendant stretching of the tense and sensitive tissue in which this occurs occasions the pain constituting "earache."

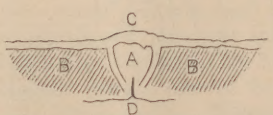
FIG. 4.



A, tooth in bone socket.
B, jaw.
C, gum, soft, not inflamed or swollen.
D, dental nerves.

Incidentally to the discussion of this subject of "dental earache," it does not seem entirely out of place to say a few words about gum lancing —a procedure which in former years was so universal, and at the present time is discountenanced to an equal degree by certain writers of authority in pediatrics.

FIG. 5.



A, tooth in bone socket.
B, jaw bone.
C, gum, tense, inflamed, swollen.
D, dental nerves.

During the dental periods two classes of irritation are met with: (1) Irritation of the dental nerves from pressure in the bone socket caused by pressure at the root of the growing tooth, and the accompanying

symptoms commonly spoken of as "teething;" in this class of cases we especially have the reflex earache spoken of above. (2) Irritation of the gum over the crown of the tooth from pressure, with symptoms of local irritation causing fever, and with general nervous symptoms.

Now we have here two exactly opposite conditions:

1. Pain from pressure at root of growing tooth.
2. Pain from pressure at crown of growing tooth.

To relieve the symptoms of (1), we can easily see that lancing the flat, soft, unirritated gum C, in Fig. 4, can be of no possible use; while what better treatment can there be for the symptoms of (2) than freely to lance the hot, swollen, and tense gum C, in Fig. 5, as we would where it is possible in any part of the economy to relieve symptoms of pressure by abolishing the cause of the pressure? Lance, then, not as formerly in every case, but knowingly in the class of cases in which our common sense tells us that the gum is crying out for the lancet as plainly as the felon demands the bistoury.

The relationship of superficial cutaneous disturbances of the face to congestions of the upper portion of the membrana tympani and tympanic cavity, as is sometimes observed in the facial eruption of measles and in the cases of which the following is a type, is somewhat complex, the arterial supply of the tympanic cavity being derived from various vessels. The anterior and middle portions are supplied: 1, by branches of the ascending pharyngeal artery (from the external carotid); 2, by branches of the middle meningeal artery which penetrate through the hiatus canalis Fallopiæ and the fissuræ petroso squamoso into the tympanic cavity; and, 3, by the internal carotid which sends branches through the bony walls of its canal.

In the case in question, there was evidently a rapid congestion of the middle ear, with no other readily assignable cause than the one mentioned, and the mechanism of the suspense of vasomotor inhibition in this and similar cases, is a question which it is intended to make the subject of further investigation.

A stout, healthy man, about thirty years of age, without history or evidence of previous ear-trouble, was exposed for an hour or more while driving to a cold, raw wind in winter; his body, head, and face were thoroughly protected, with the exception of the right side of the face and cheek, which surfaces were, of necessity, continuously exposed.

On entering the house a sense of fulness was experienced in the right ear, followed, at the end of an hour in a warm room, by a slight pain, which, by the end of another hour, had become very severe. At this period examination of the ear showed the membrana tympani congested and swollen above, but transparent below, and bulging outward; paracentesis of the membrana tympani in the posterior inferior quadrant liberated, not fluid, but air, which escaped with a sharp hiss, and was

followed, within another hour, by a serous fluid, the outflow of which rapidly increased for a time and then continued, gradually decreasing, for three days.

The fact that the bulging outward of the membrana tympani was due to compressed air, and not to accumulated fluid, as was shown by the paracentesis, is a marked symptom in this case, and was probably due to rapid swelling of the mucous membrane, first closing the Eustachian tube and then encroaching upon the lumen of the tympanic cavity sufficiently to compress the imprisoned air, and force the membrana tympani outward, and would imply that the first impulse to congestion occurred in the Eustachian tube and anterior portion of the tympanic cavity. If this conclusion is correct, the mechanism of the suspended vasomotor inhibition would be explained by the distribution of the superior maxillary nerve, which, with Meckel's ganglion, supplies the integument above and over the malar bone and that of the lower eyelid, side of the nose, and upper lip; while the connection with the ascending pharyngeal, tympanic branch, may be supplied, either by the pharyngeal nerve, which is sometimes given off from the ganglia, and sometimes directly from the Vidian, or by the small nasal branches of the Vidian which supply the membrane of the Eustachian tube.

The possible reverse train of symptoms to those of this case is presented in the following instance of reflex irritation: A boy, eight years old, who, from time to time, had had inflammation of the middle ear, but who for some months had been free from ear-trouble, after being out in the morning (the day was mild) came home with the right side of his face swollen; the swelling, which was neither red nor painful, and was not accompanied by fever, also affected the right eye, and extended over to the left eye; careful examination failed to detect any lesion to account for the symptoms, either in the mouth or elsewhere. Unfortunately the ear was not examined at this time.

In the night the child complained of slight pain in the right ear, followed in a few hours by a mucopurulent discharge, and by morning the swelling of the face had entirely disappeared, and there was a well-established otorrhœa.

In this connection we must now speak of an exceedingly important class of cases represented by the exanthemata, as here, also, there appears to be a reflex relationship between the face and the ear.

The implication of the middle ear in scarlet fever, for instance, occurs in two distinct ways: the first, and by far the most frequent, is that in which an inflammatory process in the middle ear is the secondary result of the inflammation of the mucous membrane of the fauces and nasopharynx, the first step in the mechanism of the aural implication being a more or less general congestion of the tympanic mucous membrane, blood stasis, due to the swelling of the mucous membrane of the Eus-

tachian tube and the consequent closure of that passage, a condition which permits the rarefaction of the air in the tympanic cavity, and consequently still further favors congestion in addition to the interference with the venous circulation.

In the second instance the primary congestion occurs principally in the upper portion of the tympanic cavity and membrana tympani, and is coincident with the facial eruption, a sequence explainable on the reflex hypothesis; the same sequence is still more frequently observable in the facial eruption of measles.

An illustration of this second instance was beautifully represented in a patient coming under our notice with an intense efflorescence of measles on the face and no aural symptoms; on examining the ears, however, a marked congestion was found in the upper part of the membranæ tympani.

The next and last case of our series represents a rather more extended line of reflex symptoms, and though resembling some of the other cases, is interesting as presenting a combination of reflex phenomena from the face and throat to the ear and thence to the lung.

A girl, aged nineteen months, well until March 6th, when she began to have cough, coryza, and lachrymation; these symptoms continued on the 7th, and an efflorescence of measles declared itself on the face. Temperature 104½° F., pulse 150, respiration 40; on the 8th there was no extension of the efflorescence, and at 4.30 P.M. there was a general eclamptic attack, but consciousness returned at 5 P.M., cough less and apparently caused by a catarrhal condition of the pharynx and larynx, lungs normal, efflorescence appearing on body and limbs; in the night great restlessness and apparently pain, which, however, could not be localized, though it was suspected to be in the chest, as the respirations began to increase and to be of a short grunting character; these symptoms continued until at 5 A.M., March 9th, the respirations numbered 70 to 80 in a minute, and the picture was that of a pneumonia; frequent auscultation and percussion revealed nothing abnormal in the lungs or heart; at 6 A.M. a slight mucopurulent discharge was noticed on the pillow, coming from the left ear; the temperature was then found to be a degree lower, but the rapid breathing continued. The left Eustachian tube was now inflated, and in about ten minutes the respiration was almost normal; in the further progress of the disease, the measles ran a normal course, but the right ear was also affected and both ears continued to discharge. A number of times the rapid respiration returned, and was always promptly relieved by the use of the air douche.

Rational treatment of these cases, then, can only be accomplished by a thoroughly scientific anatomical and physiological knowledge of the reflex phenomena, which, from the hypersensitive condition of the child's

nervous system, play a much greater *role* in childhood than in adult life and render a diagnosis much more difficult.

Finally, from what has been said, we must admit the great importance of recognizing whether symptoms are real or reflex, tracing them from one end of the labyrinth of nerves to the other, and following them out with the minutest detail, for in this way only can we speedily reach the goal of recovery through definite and exact diagnosis, with resulting appropriate prognosis and treatment.

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706 & 708 Sansom Street, Philadelphia.